sis[1] is known to arise as a paired epiblastic involution (Dohrn). In the Cyclostomata it is formed as an epiblastic involution (possibly paired) at the extreme anterior end of the body. In one Vertebrate alone, Myxine (*vide* figure), it still opens into the hypoblast; in all others it approaches the hypoblast in development, but does not fuse with that layer. It always lies in very close relationship with the extreme end of the notochord—that is, with the end of a structure derived from the hypoblast.

In adult Petromyzon, in which the tube of the oral hypophysis has the same relationships as in Myxine, except that the posterior opening into the hypoblastic sac is absent, it nevertheless has an astonishing length, and ends blindly very close to the gut. In Myxine and Petromyzon, tubular glands are developed in connection with it. In all the higher Vertebrates, in which the oral part is very rudimentary, it always has a distinct glandular character.

And now, what of the last condition? This also is satisfactorily met. In all cases the oral hypophysis has a special, and indeed large, process of nervous matter (the processus infundibuli, or neural hypophysis), which is derived from the posterior part of the fore-brain, from the base of the infundibulum. This process is concerned with the innervation of the oral hypophysis alone. In Myxine and Petromyzon alone, so far as my researches extend (possibly also in Protopterus), this nervous system is not rudimentary. In most Vertebrates the neural hypophysis, which, as Kölliker aptly remarks, is at first composed of the same cell elements and fibres as the rest of the brain, degenerates, and in very many full-grown animals forms a mass of tissue, the structure of which many observers have compared to that of the suprarenal bodies (known to be masses of degenerated tissue).

The neural hypophysis is thus the most remarkable structure in the whole of the Vertebrate central nervous system. Though degenerated, it still clings to the traditions of its ancestry, for even, as it were, in its death it is closely and almost inseparably connected with the rest of the hypophysis, especially in Mammalia and in Dipnoi. In Myxine alone, of all Vertebrates, the old mouth still retains some of its functions as a mouth; it conducts the water of respiration to the gills. In this case, even, changes have occurred, for the nose[2] (see figure, *n.f.*) has got partly involved in the passage of the old mouth. If it be true that the nose was originally a branchial sense-organ—which view, in spite of Gegenbaur, I still maintain—its assumption of a position in the passage of the old mouth in Myxine is, on purely physiological grounds, intelligible.

It is well known that that which I call the old mouth in Myxine is purely respiratory, conducting water into the gills; and what then could be more likely than that one of the branchial sense-organs should be, as it were, told off to do duty at its entrance. It is certain, from Goette's and Dohrn's researches, that these passages in Myxine and Petromyzon are the representatives of the oral hypophysis. I have gone over and extended these observations, and can fully confirm Dohrn in nearly every point, and all I claim here is the identification of the hypoblastic opening in Myxine as the (modified) opening of the old mouth into the gut.

If the above morphological comparison can be maintained (and I believe it can), the importance of its bearing on the morphology of Vertebrates can hardly be over-estimated.

A number of other problems and conclusions arise out of all this, but I reserve the consideration of these for a much more exhaustive work, in which the literature of the subject will receive full attention. J. BEARD.

Anatomisches Institut, Freiburg i/B., November 16.

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[1] I believe it is very frequently paired, though not at its point of origin.

[2] In Petromyzon, Dohrn finds that the nose is at first a special depression apart from the hypophysis invagination. The latter lies between the nose and mouth.

TIMBER, AND SOME OF ITS DISEASES.[1]

III.

HAVING now obtained some idea of the principal points in the structure and varieties of normal healthy timber, we may pass to the consideration of some of the diseases which affect it. The subject seems to fall very naturally into two convenient divisions, if we agree to treat of (1) those diseases which make their appearance in the living trees, and (2) those which are only found to affect dead timber after it is felled and sawn up. In reality, however, this mode of dividing the subject is purely arbitrary, and the two categories of diseases are linked together by all possible gradations.

Confining our attention for the present to the diseases of standing timber—*i.e.* which affect undoubtedly living trees—it can soon be shown that they are very numerous and varied in kind; hence it will be necessary to make some choice of what can best be described in this article. I shall therefore propose for the present to leave out of account those diseases which do injury to timber indirectly, such as leaf-diseases, the diseases of buds, growing roots, and so forth, as well as those which do harm in anticipation by injuring or destroying seedlings and young plants. The present article will thus be devoted to some of the diseases which attack the timber in the trees which are still standing; and as those caused by fungus parasites are the most interesting, we will for the present confine our attention to them.

It has long been known to planters and foresters that trees become rotten at the core, and even hollow, at all ages and in all kinds of situations, and that in many cases the first obvious signs that anything is the matter with the timber make their appearance when, after a high gale, a large limb snaps off, and the wood is found to be decayed internally. Now it is by no means implied that this rotting at the core—"wet-rot," "red-rot," &c., are other names generally applied to what is really a class of diseases—is *always* referable to a single cause; but it is certain that in a large number of cases it is due to the ravages of fungus parasites. The chief reason for popular misconceptions regarding these points is want of accurate knowledge of the structure and functions of wood on the one hand, and of the nature and biology of fungi on the other. The words disease, parasitism, decomposition, &c., convey very little meaning unless the student has had opportunities of obtaining some such knowledge of the biology of plants as can only be got in a modern laboratory: under this disadvantage the reader may not always grasp the full significance of what follows, but it will be at least clear that such fungi demand attention as serious enemies of our timber.

It will be advantageous to join the remarks I have to make to a part description of some of the contents of what is perhaps one of the most instructive and remarkable museums in the world—the Museum of Forest Botany in Münich, which I have lately had the good fortune to examine under the guidance of Prof. Robert Hartig, the distinguished botanist to whose energy the Museum is due, and to whose brilliant investigations we owe nearly all that has been discovered of the diseases of trees caused by the Hymenomycetes. Not only is Prof. Hartig's collection unique in itself, but the objects are classical, and illustrate facts which are as yet hardly known outside the small circle of specialists who have devoted themselves to such studies as are here referred to.

One of the most disastrous of the fungi which attack living trees is *Trametes radiciperda* (Hartig), the *Polyporus annosus* of Fries, and it is especially destructive to the Coniferæ. Almost everyone is familiar with some of our common Polyporei, especially those the fructifications of which project like irregular brackets of various colours from dead stumps, or from the stems of moribund trees;

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[1] Continued from p. 207.

well, such forms will be found on examination to have numerous minute pores on the under side or on the upper side of their cheese-like, corky, or woody substance, and the spores which reproduce the fungus are developed on the walls lining these many pores, to which these fungi owe their name. *Trametes radiciperda* is one of those forms which has its pores on the upper side of the spore-bearing fructification, and presents the remarkable peculiarity of developing the latter on the exterior of roots beneath the surface of the soil (Fig. 11).

This is not the place to discuss the characters of species and genera, nor to enter at any detail into the structure of fungi, but it is necessary to point out that in those cases where the casual observer sees only the fructification of a Polyporus, or of a toadstool, or of a mushroom (projecting from a rotting stump or from the ground, for instance), the botanist knows that this fructification is attached to, and has taken origin from, a number of fine colourless filaments woven into a felt-like mass known as the mycelium, and that this felt-work of mycelium is spreading on and in the rotten wood, or soil, or whatever else the fungus grows on, and acts as roots, &c., for the benefit of the fructification.

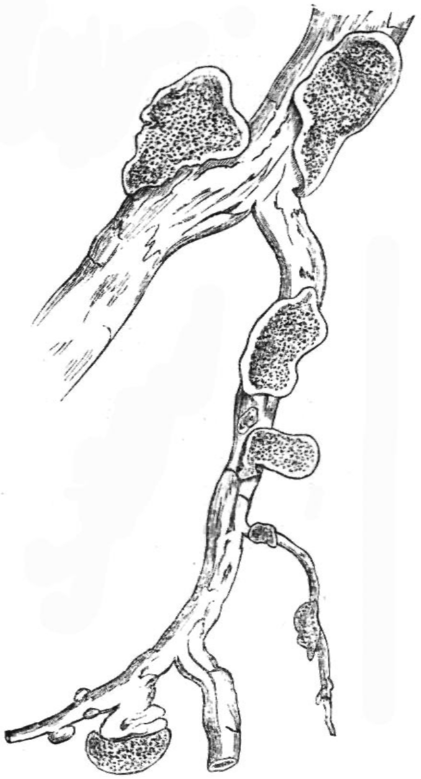

FIG. 11.—Portion of root of a spruce-fir, with fructification of *Trametes radiciperda* (after Hartig). Each fructification is a yellowish-white mass of felt-like substance spread over the root, and with minute pores, in which the spores are produced, on its outer surface; the mycelium which has developed it is in the interior of the root.

Now, the peculiarity of the mycelium of this *Trametes radiciperda* is that it spreads in the wood of the roots and trunks of pines and firs and other Conifers, and takes its nourishment from the wood-substance, &c., and it is to the researches of Hartig that we owe our knowledge of how it gets there and what it does when there. He found that the spores germinate easily in the moisture around the roots, and put forth filaments which enter between the bark-scales, and thus the mycelium establishes itself in the living tree, between the cortex and the wood (Fig 12). It is curious to note that the spores may be carried from place to place by mice and other burrowing animals, since this Trametes is apt to develop its fructification and spores in the burrows, and they are rubbed off into the fur of the animals as they pass over and under the spore-bearing mass.

When the mycelium obtains a hold in the root, it soon spreads between the cortex and the wood, feeding upon, and of course destroying, the cambium. Here it spreads in the form of thin flattened bands, with a silky lustre, making its way up the root to the base of the stem, whence it goes on spreading further up into the trunk (Fig. 12).

Even if the mycelium confined its ravages to the cambial region, it is obvious, from what was described in Articles I. and II., that it would be disastrous to the tree; but its destructive influence extends much further than this. In the first place, it can spread to another root belonging to another tree, if the latter comes in contact in the moist soil with a root already infected; in the second place, the mycelium sends fine filaments in all directions into the wood itself, and the destructive action of these filaments—called hyphæ—soon reduces the timber, for several yards up the trunk, to a rotting, useless mass. After thus destroying the roots and lower parts of the tree, the mycelium may then begin to break through the dead bark, and again form the fructifications referred to.

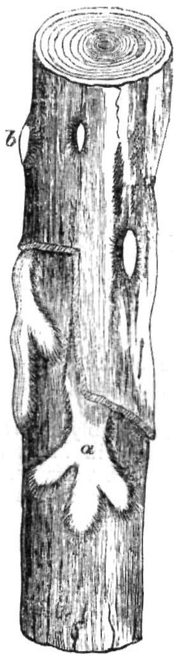

FIG. 12.—Piece of root of spruce-fir, with the mycelium of *Trametes radiciperda* (after Hartig) enlarged about 3 times. The white mycelium spreads in a fan-like manner over the surface beneath the cortex, as seen in the figure where the latter has been lifted and removed (*a*). Here and there the mycelium bursts through the cortex in the form of white protuberances (*b*), to form the fructifications.

Since, as we shall see, *Trametes radiciperda* is not the only fungus which brings about the destruction of standing timber from the roots upwards, it may be well to see what characters enable us to distinguish the disease thus induced, in the absence of the fructification.

The most obvious external symptoms of the disease in a plantation, &c., are: the leaves turn pale, and then yellow, and die off; then the lower part of the stem begins to die, and rots, though the bark higher up may preserve its normal appearance. If the bark is removed from one of the diseased roots or stems, there may be seen the flat, silky, white bands of mycelium running in the plane of the cambium, and here and there protruding tiny white cushions between the scales of the bark (Fig. 12); in advanced stages the fructifications developed from these cushions may also be found. The wood inside the diseased root will be soft and damp, and in a more or less advanced stage of decomposition.

On examining the timber itself, we again obtain distinctive characters which enable the expert to detect the disease at a glance. I had the good fortune to spend several pleasant hours in the Münich Museum examining and comparing the various diseases of timbers, and it is astonishing how well marked the symptoms are. In the present case the wood at a certain stage presents the appearance represented in the drawing, Fig. 13. The general tone is yellow, passing into a browner hue. Scattered here and there in this ground-work of still sounder wood are peculiar oval or irregular patches of snowy white, and in the centre of each white patch is a black speck. Nothing surprised me more than the accuracy with which Prof. Hartig's figures reproduce the characteristic appearance of the original specimens in his classical collection, and I have tried to copy this in the woodcut, but of course the want of colour makes itself evident.

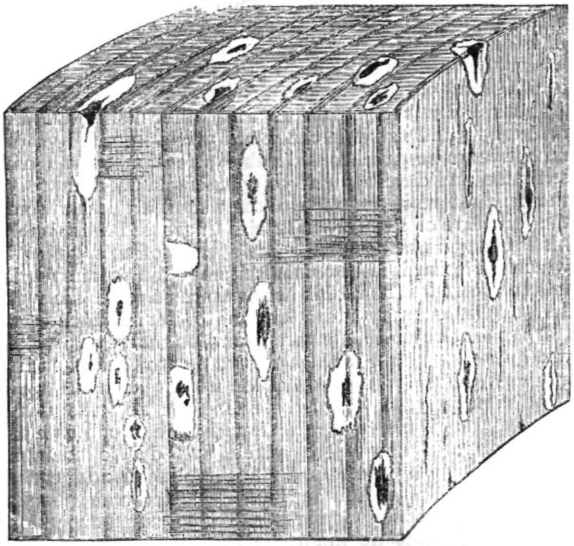

Fig. 13.—A block of the timber of a spruce-fir, attacked by *Trametes radiciperda*. The general colour is yellow, and in the yellow matrix of less rotten wood are soft white patches, each with a black speck in it. These patches are portions completely disorganized by the action of the mycelium, and the appearance is very characteristic of this particular disease. (After Hartig.)

It is interesting and important to trace the earlier changes in the diseased timber. When the filaments of the fungus first begin to enter the wood, they grow upwards more rapidly than across the grain, piercing the walls of the cells and tracheides by means of a secretion—a soluble ferment—which they exude. This ferment softens and dissolves the substance of the walls, and therefore, of course, destroys the structure and firmness, &c., of the timber. Supposing the filaments to enter cells which still contain protoplasm and starch, and other nutritive substances (such as occur in the medullary rays, for example), the filaments kill the living contents and feed on them. The result is that what remains unconsumed acquires a darker colour, and this makes itself visible in the mass to the unaided eye as a rosy or purple hue, gradually spreading through the attacked timber. As the destructive action of the fungus proceeds in the wood, the purple shades are gradually replaced by a yellowish cast, and a series of minute black dots make their appearance here and there; then the black dots gradually surround themselves with the white areas, and we have the stage shown in Fig. 13.

These white areas are the remains of the elements of the wood which have already been completely delignified by the action of the ferment secreted by the fungus filaments—*i.e.* the hard woody cell-walls have become converted into soft and swelling cellulose, and the filaments are dissolving and feeding upon the latter (Fig. 14). In the next stage of the advancing destruction of the timber the black dots mostly disappear, and the white areas get larger; then the middle-lamella between the contiguous elements of the wood becomes dissolved, and soft places and cavities are produced, causing the previously firm timber to become spongy and soft, and it eventually breaks up into a rotting mass of vegetable remains.

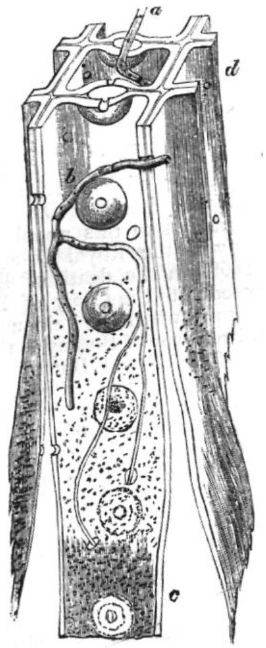

Fig. 14.—Sectional view of a tracheide of the spruce-fir, attacked by the hyphæ (*a*, *b*) of a *Trametes*, highly magnified (after Hartig). The upper part of the tracheide has its walls still sound, though already pierced by the hyphæ; the lower part (*c*) has the walls completely delignified, and converted into cellulose, which swells up and dissolves. The middle-lamella is also undergoing dissolution. The holes in the walls have been bored by hyphæ.

It will readily be understood that all these progressive changes are accompanied by a decrease in the specific gravity of the timber, for the fungus decomposes the substance much in the same way as it is decomposed by putrefaction or combustion, *i.e.* it causes the burning off of the carbon, hydrogen, and nitrogen, in the presence of oxygen, to carbon-dioxide, water, and ammonia, retaining part in its own substance for the time being, and living at its expense. H. Marshall Ward.

(*To be continued.*)

PROFESSOR ALEXANDER DICKSON.

THE close of 1887 has been marked by a long death-roll in the ranks of science. In the company of botanists it has been especially heavy, and now the sad news of the tragically sudden death of Prof. Alexander